situs transversus, and we recognize electrocardiography as a valuable adjunct in the differential diagnosis of cardiac displacements.

REFERENCES.

1. Abbott, M. E.: Congenital cardiac disease. In: Mod. Med., Philadelphia, Lippincott, 1908, iv, 323.
2. Bryce, T. H.: Embryology. In: Quain's anatomy. New York, Longmans, 1908, i, p. 63.
3. Hirschfelder, A. D.: Diseases of the heart and aorta. Philadelphia, Lippincott, 1918, p. 562.
4. Hoke, E.: Ueber das Elektrokardiogramm eines Falles von Situs viscerum inversus totalis. München. med. Wchnschr., 1911, lviii, 802.
5. Lewis, T.: Electrocardiography and its importance in the clinical examination of heart affections. Brit. Med. Jour., 1912, i, 1421–1423.
6. Neuhof, S.: Clinical cardiology. New York, Macmillan, 1917, p. 32.
7. Nicolai, G. F.: Das Elektrokardiogramm bei Dextrocardie und anderen Lageveränderungen des Herzens. Berl. klin. Wchnschr., 1911, xlviii, 51–55.
8. Owen, S. A.: A case of complete transposition of the viscera, associated with mitral stenosis; including a description of the electrocardiographic tracings. Heart, 1911–1912, iii, 113–117.

PROTOZOAL INFECTIONS OF THE INTESTINES:

WITH EMPHASIS ON THEIR INCIDENCE AND BEHAVIOR IN OTHER THAN TROPICAL REGIONS AND ON THE PATHOGENICITY AND TREATMENT OF CERTAIN OF THE FLAGELLATES.

By Robert Pollock, M.D.,

and

Rawson J. Pickard, M.D.,

San Diego, California.

In war time the environment of every military camp is menaced by the diseases that become epidemic among the troops.

That protozoal infections are not confined to the tropics we are having abundant evidence from the reported cases and studies by men in every part of the country. These parasites are of special interest at present on account of the movement of carriers, due to war activities, with the infection of new regions and new contacts. In the Philippines and in the Canal Zone amebic dysentery was one of the foremost foes of white men, soldiers or civilians. Sanitation and preventive medicine are doing much to stop the spread of this and of other protozoal infections, but they must still be looked upon as dangers to camp life and to the adjacent communities.

This is particularly true now that we know of the existence of carriers who need never have visited in Southern countries and in whom the infection seldom declares itself in a frank dysentery, so that they go about for years improperly treated unless their physician is exceptionally thorough in his investigations.

The indications for fecal study were outlined by Sistrunk[1] in his paper, one of the early reports of so-called tropical infections occurring in residents of other regions. The stools of 110 patients with either chronic diarrhea, the passage of blood and mucus, anemia or obscure abdominal symptoms were examined. Of these, 65 had intestinal protozoa, chiefly amebas or trichomonas.

Smithies[2] reported in July, 1917, the study of the stools of 1000 patients at Augustana Hospital, in which series 98, or 9.8 per cent., showed intestinal protozoa either ameba or one of the flagellates. This report with others has undoubtedly quickened our interest in the subject and made us keener in searching for and recognizing the protozoa even though occurring far from the tropics. Practically all of Dr. Smithies' cases came from the northern half of the Central States. It is worthy of note that in most of his cases two or more varieties of protozoa were found in the stools.

It may not be out of place here briefly to summarize our knowledge of the habits of the parasitic protozoa and the principles governing their control.

Entamebic dysentery is caused by a distinct species of ameba (ameba histolytica), an obligatory parasite, whose host, so far as is known definitely, is man. The disease can reach the intestine of man only by the ingestion of material containing Entameba histolytica, generally in the encysted stage. These ameba may originate from acute cases or, more commonly, from convalescent carriers, or even from individuals who have never had dysentery, but are contact carriers. Such apparently healthy carriers on account of the cysts in their stools are more dangerous than the patient in the acute stage of the disease.

The flagellates (cercomonas, trichomonas, tetramitus) have not until recently been generally regarded as pathogenic. For this reason they have not been so thoroughly studied nor have means to combat them been so much searched for. In Panama the chief interest they called forth was the frequency with which they were associated with ameba, demanding more careful search for the latter. Trichomonas, the one most commonly found, does not form cysts so far as known, the so-called cysts being blastocystis hominis.

In regard to the flagellates, Brumpt[3] says that diarrheal conditions have been attributed to them when present in large numbers. A certain amount of irritation is necessary for their development, but once started, their multiplication may keep up a chronic intestinal catarrh. In the Andaman Islands, Anderson found trichomonas 723 times in 920 cases of dysentery, associated with ameba in 459 cases; flagellates were rare in 210 non-dysentery patients. Brumpt says that patients with monads almost always have a low or absent stomach acidity.

---

[1] Jour. Am. Med. Assn.. November 4, 1911, No. 19, lvii, 1507.

[2] Medicine and Surgery, July, 1917, p. 400.

[3] Parasitologie, Paris, 1913.

The attitude of the military surgeon to the protozoa is largely one of prophylaxis. Once having gained entrance to a camp, they tend rapidly to become epidemic and threaten to incapacitate large numbers of men. Such conditions call for the most intelligent and vigorous prophylactic measures, as shown by our experience in the Philippines, the Canal Zone and on the Mexican border.

The attitude of the civilian physician to the sporadic cases he meets is more one of the therapeutic handling of the individual case, although the protection of those about the patient ought not to be overlooked simply because it presents comparatively little difficulty.

The question of prophylaxis is essentially that in typhoid fever. The details of these prophylactic measures are so thoroughly standardized that we shall not enter into their discussion. As to the specific destruction of the intruder there is still much variation of opinion and the therapeutic treatment continues to tax the best efforts of clinicians. One after another, specific methods of treatment have been proclaimed as successful, only to give place later to a new method boasting a higher percentage of cures. Just what constitutes a permanent cure seems still a vexed question with certain authors. May not the difference in susceptibility of protozoa to chemicals, shown by Brem and Zeiler[4] in experimenting on ameba with quinin, and as suggested by them in reference to ipecac, determine to some extent the time demanded for permanent cure, as well as the selection of the chemical for the individual case?

A few facts related to treatment stand forth as apparently proved. Ipecac in some form seems to have a distinctly beneficial effect upon the symptoms and plays at least a part in practically all methods of treatment directed against the ameba, although upon the flagellates it seems to exert only temporary influence. It would seem that in the attack upon the ameba it is necessary to place the drug both in the blood stream and on the intestinal lining, although some clinicians (Manson, Dock, Niles and Simon) claim satisfactory results from the use of large doses of the powdered drug placed below the stomach by means of properly protected pills, a good description of which is given by Niles.[5] Others use the alkaloid emetin placed into the circulation by needle, sometimes supplemented by arsenic subcutaneously or intravenously. The advocates of both of these methods claim good results and see no need of colonic irrigations as an aid to the specific treatment.

It seems to be becoming more and more popular to attack every known or suspected protozoal infection, from pox to pellagra, by pumping arsenic into the circulation. We are, however, impressed with the results claimed by certain observers from the combined use of emetin hypodermically and salvarsan intravenously. The use of arsphenamin and neosarsphenamin has been extended to the amebic infections by Wynne,[6] and is now established as a definite treatment when used in connection with emetin. The recent paper by Dr. Herbert Gunn[7] at the meeting of the California State Medical Society is a valuable contribution to the treatment of carriers.

---

[4] Am. Jour. Med. Sc., November, 1910, p. 669.

[5] Ibid., October, 1914, p. 526.

While touching briefly on treatment we cannot resist calling attention to the importance of detecting and subjecting to treatment the carriers of the Entameba histolytica. They are doubly dangerous in any community up to the time that suspicion is directed toward them, after which a careful study of the stool promptly identifies them, and they are then fair targets for therapeutic attack. These carriers present the ameba in the encysted form, which apparently is impervious to ordinary attack. An agent interesting to clinicians in this connection is the emetin-bismuth-iodide, which by some recent observers has seemed to be at least fairly satisfactory against encysted ameba. It is not claimed to have any appreciable effect upon flagellates. We feel also impelled to record the only method that with us has proved at all satisfactory in treating the flagellate trichomonas intestinalis, namely, the giving of large doses of calomel followed by saline purgation, once every four or five days, coupled with daily colonic irrigation of mercuric chloride (1 in 10,000 to 1 in 20,000). We are experimenting with other chemicals in the hope of finding something that will have a specific and permanent effect on them.

In a group of 46 cases coming under our notice during the past year, with symptoms seeming to demand a careful search for intestinal parasites, 19, or 41.3 per cent., showed protozoal parasites many times accompanied by other intruders. So frequently have the trichomonas presented, in association with troublesome and even grave symptoms, that we incline to agree with Freund,[8] who says: "I firmly believe that trichomonas is the direct cause of certain forms of acute, chronic and recurring enteritis, with abdominal pain, diarrhea, loss in weight, elevation of temperature and at times anemia." At the time (1908) when he made this statement he reported one case that died of progressive pernicious anemia and another less grave that recovered, although strongly suggesting the pernicious type of anemia.

While our group is too small to serve as a basis for statistical analysis, yet the suggested prevalence of these diseases in our community is worthy of attention. Also, the little group presents so many cases of striking individual interest, both as to course and treatment, that we have felt justified in discussing a few of them.

---

[6] Cited by James: Proc. Med. Assn., Canal Zone, vol. vi, part 1, p. 98.

[7] California State Jour Med., May, 1918, p. 240.

[8] Arch. Int. Med., 1908, No. 1, i, 28.

CASE I.—A mining engineer, aged thirty-six years, contracted amebic infection in Colombia and had been cured and had relapsed several times before coming under our notice. He was emaciated, anemic and running a temperature ranging from 99° to 102°. He was sensitive to pressure throughout the entire abdomen and had griping pain when the bowel moved. The stools were frequent and liquid, containing mucus, pus and blood, and showed under the microscope myriads of Ameba histolytica. This case presented the clinical picture of a severe amebic infection. The outlook was not promising and the progress at first was discouraging. However, with absolute rest in bed and careful attention to diet and nursing, he made a good recovery in about three months. He returned to the city about six months later, with a distinct relapse to all of his old symptoms, although they were not so severe. He was again treated much as before, with similar results, and after nine months has not again relapsed. This case we feel illustrates a not uncommon course of severe amebic infection treated in the most generally accepted manner, with emetin injections, ipecac by mouth and colonic saline irrigation. He relapsed because the encysted ameba were not all destroyed, and meanwhile he was a convalescent carrier of constant menace to those about him.

Case No.	Reported in detail.	Sex.	Age.	Place of original infection.	Clinical symptoms.	Protozoa.
1	r[1]	M.	26	South America	Dysentery	Ameba histolytica.
2	r	M.	23	Mexico	Dysentery	Ameba histolytica.
3	r	M.	43	South America	Hyperchlorhydria	Ameba histolytica; oxyuris; bothriocephalus; round-worm.
4	..	F.	35	United States	Dysentery	Ameba histolytica; trichomonas.
5	..	M.	65	......	Achylia; diarrhea	Trichomonas.
6	r	M.	47	Philippines	Nervous exhaustion; hypochlorhydria	Ameba histolytica; trichomonas.
7	r	F.	43	Minnesota	Diarrhea; hyperchlorhydria	Trichomonas.
8	r	F.	47	Mexico	Fever; chills	Trichomonas.
9	..	F.	46	......	....	Trichomonas.
10	r	M.	68	Wisconsin	Anemia (pernicious?)	Ameba histolytica; trichocephalus.
11	r	M.	38	Mid-West	Intermittent diarrhea	Ameba histolytica: trichomonas.
12	..	M.	50	South Africa	Intestinal indigestion; constipation	Ameba histolytica.
13	..	M.	59	Mexico	Dysentery	Ameba histolytica; uncinaria; strongyloids.
14	..	M.	58	New Jersey	Dysentery	Ameba histolytica.
15	..	M.	40	North Carolina	Dysentery	Ameba histolytica.
16	..	F.	40	Nebraska	Constipation	Ameba histolytica and monads.
17	r	M.	37	Cuba	Attacks of somnolence	Ameba histolytica; trichomonas.
18	..	F.	65	Japan	Diarrhea; achylia	Ameba histolytica.
19	r	M.	43	Northern U. S.	Diarrhea	Trichomonas.

[1] r = reported at length in body of paper.

Case II.—Male, Spaniard, aged twenty-three years, who had lived in Mexico, presented typical symptoms of acute amebic dysentery; acquired complete relief of symptoms under intravenous injections of emetin and kerosene enemata. Like Case I, the cysts persisted in the stools and were so recorded when he returned to Mexico against our advice.

Case III.—A mining prospector, aged forty-three years, who had suffered severe malaria, with intestinal hemorrhages, while in Colombia, on his return to California sought relief for definite hyperchlorhydria symptoms. The blood examination, while showing no anemia, directed our attention to the bowel by a 7 per cent. eosinophilia. The stools on examination showed pin-worms, many ova of Bothriocephalus latus, an unknown species of round-worm and numerous Ameba histolytica. This man has had no dysentery since his malaria, and after his hyperchlorhydria was corrected by proper diet, he expressed himself as feeling well, and shortly afterward disappeared. He had been cleaned out with calomel and santonin and given a course of alcresta ipecac, with what permanent results we are unable to report. The case is quoted to illustrate the effect of intestinal intruders upon the blood and stomach glands as well as to show that a strong adult can carry about quite a lively fauna, with none of their characteristic symptoms in evidence.

Case VI.—An army officer, aged forty-seven years, who had seen service in the Philippines, suffering both severe malaria and yellow fever, but not dysentery. Repeated stool examinations during the previous year had always shown trichomonas intestinalis hominis. He was nervous and generally run down, with a slight degree of anemia and a lowered gastric chemistry. The bowel was for the most part slightly constipated; abundant monads were found in the stools. He was persuaded to start his furlough with several weeks' rest and upbuilding treatment. Although ameba later showed in the stools, he markedly gained in every way, and is now at the end of a year actively engaged on the Western front. We were unable to learn how completely the protozoa were eliminated from the stools at the army hospital where he was later treated, but we feel that his transference to Northern latitudes would have a tendency to reduce their virulence. This case suggests the possibility of overlooking the ameba even when attention is directed to the stools by the presence of other parasites.

Case VII.—An unmarried woman, aged forty-three years, teacher and housekeeper, from Minnesota, never had been in a tropical latitude. Complained of hyperchlorhydria symptoms and tendency to diarrhea for many years. The hyperchlorhydria was demonstrated by analysis, with some pylorospasm and a ptosed stomach. The stools contained monads but no ameba. Careful roentgen-ray study showed no evidence of ulcer. Her stomach symptoms gradually improved under proper diet and support of that viscus. Under this treatment and intermittent courses of colonic flushings a normal physiological bowel function was established without any tendency to loose stools. We are inclined to look upon this case as one of chronic diarrhea, due to monad irritation.

CASE VIII.—A somewhat analogous case was an unmarried woman, aged forty-seven years. She had never been of robust physique, although in no sense an invalid. For the past ten years, dating from a visit to Southern New Mexico, she had presented occasional fever paroxysms, much like a malarial seizure, with marked chill followed by 1° to 4° of fever and marked sweating. This complete cycle would be repeated daily for three to five days and gradually disappear. A blood examination made during a chill showed a leukocytosis of 17,000, with a straight polynuclear advance to 95 per cent. No malarial plasmodia could be demonstrated at any of several blood examinations. Slight evidence of colitis, without any bowel looseness, directed attention to the stools, which proved on examination to be swarming with monads. In this case the monads could be made to disappear from the stools by the free use of calomel by mouth and mercuric chloride (1 to 20,000) flushings of the colon. After some months of observation we found that if the monads were allowed to return to the stools the patient would promptly suffer a return of her paroxysms, which were absent as long as the stools were monad-free. This patient is still under observation. Her stomach chemistry was that of a moderate hypochlorhydria, with a tendency to constipation. Methylene blue and thymol were tried without success in this case.

CASE X.—An American business man, still active at sixty-eight, a year ago applied for relief from obstinate diarrhea. He showed complete absence of HCl and ferments in the gastric juice, and his diarrhea for a time was relieved by suitable diet and the free use of dilute HCl. A few months later he became quite anemic and dyspneic and his bowel began to trouble him again. The blood showed a marked secondary anemia, obscurely close to pernicious in type, the red cells falling to 1,500,000, and with the color index constantly 1.5. The stool showed abundant Ameba histolytica and trichomonas. The kidneys and other organs except the stomach were negative. By keeping him at rest and controlling his diet this patient's blood was gradually built up to normal in about three months, cacodylate of iron being used liberally in deep gluteal injections. This may have helped in the fight against the ameba, although both emetin hydrochloride and ipecac in pill form were persistently used. In this case we are inclined to attribute the final disappearance of the ameba to the free use of salol-coated pills of powdered ipecac. A few monads have persisted in this case, more, we feel, because of the patient's refusal to be "fussed" with when once he felt well again. This man had never been in the tropics.

Case XI.—An energetic American business man, aged thirty-eight years, complained of having had intermittent diarrhea most of his life, as had his father before him. He came from one of the Northern Middle West States and had never been out of the United States. His mild stomach symptoms were the expression of a moderate hypochlorhydria. The stools showed both Ameba histolytica and trichomonas in abundance. As he refused to rest he was treated throughout as an ambulatory case. He presented but little anemia and was in fair flesh and strength. Hypodermics of emetin followed by "Al Cresta" tablets cleared up the ameba, which were presumably not a very virulent strain. The monads also cleared up under the mercury treatment previously mentioned. Most of the monads we have met seemed to thrive in solution of methylene blue. At last reports about two months ago this case was apparently well. When compared with Case I it suggests the variability of virulence of these protozoa.

Case XVII.—An American male, aged thirty-seven years, who had seen military service in Cuba and suffered from an epidemic of ileocolitis while encamped in Florida. He complained chiefly of having had for eighteen years lethargy, during attacks of which he would practically fall asleep while going about. The attacks would come on while working, riding horseback, talking, etc., and were fought off with great difficulty. Ameba histolytica and trichomonas were found in the stools. His somnolent attacks cleared up under emetin. Mild attacks recurred after three months and emetin was resorted to again and controlled the recurrence of his lethargic feeling. Two or three doses of the drug usually suffice for this purpose.

Case XIX.—An American business man, aged forty-three years, who gave a history of recurring diarrheas extending over several years, attendant upon a general nervous breakdown. He had multiple diverticulitis, with general peritonitis and repeated abdominal abscesses. This case ran a long tedious course of abdominal trouble, with several operations to drain abscesses. As monads were early noticed in the stools we cannot resist speculating as to their possible role in the production of his trouble. Might not the monads have early produced an irritation which invited other infection?

In conclusion, we wish to lay stress on a few points:

1. San Diego County undoubtedly contains many cases of protozoal infection more or less active, as well as many convalescent carriers.
2. Some of these cases from Northern latitudes probably become more active by reason of the climatic change, the effect of a marked change of climate and latitude upon the virulence of protozoa having long been known.

3. All patients presenting obscure bowel symptoms, especially if anemic or achylous, should have the stools carefully searched for parasites.

4. The pathogenicity and behavior of the intestinal protozoa vary under different conditions of climate, latitude, exercise, diet and medicinal treatment.

5. The flagellates (monads) are capable of distinctly pathogenic behavior.

6. The most satisfactory treatment we have applied to the monads up to the present is the double mercury treatment above outlined.

CHRONIC JAUNDICE—FAMILY TYPE.[1]

By James I. Johnston, M.D.,

ASSISTANT PROFESSOR OF MEDICINE, UNIVERSITY OF PITTSBURGH; PHYSICIAN TO THE MERCY HOSPITAL, PITTSBURGH, PA.

Until a few years ago there was little published in regard to this type of chronic jaundice. In 1910 there was shown at the University of Toronto, to the members of the Interurban Clinical Society, a young man suffering from chronic jaundice, with enlarged spleen, which had existed since childhood, and with periods of exacerbation when the jaundice became more intense. There had been an uncle in the family who had suffered from the same condition, but neither one of them had been incapacitated in any way, and complained only of the persistent discoloration. This case shown was diagnosed as family jaundice, but no other analyses were brought to our attention. Since 1913 a number of articles have been written concerning this disease. The French writers are said to have reported on this affection more fully and elaborately than others, but now several American clinicians have reported cases. The best term to express the disease seems to be splenomegaly with or in chronic, congenital, hemolytic jaundice. The disease is exceedingly rare and its pathology is not understood. It is true jaundice and not pigmentation of the skin, such as occurs in Addison's disease or in what is known as Gaucher's disease, which is also a splenomegaly. As one of the terms of its name expresses, it is a hemolytic and not an obstructive jaundice. The disease clinically is characterized by the striking general chronic icterus, the large spleen, moderate anemia, good general health, and the possible history of more than one case in a family. The etiology of this disease is unknown, its pathology unexplained, and its entity recognition manifest by certain signs and symptoms together with blood and urine changes. There are two theories in regard to its manifest symptomatology as well as its cytologic analysis. One is that there is an imperfect formation of the blood, and the other is that the normal cells are unstable or fragile

---

[1] Read before the Pittsburgh Academy of Medicine.